ejaculated spermatozoa during coitus, Mukherjee suggests. Sperm, although they display foreign surface constituents, do not normally elicit an antibody response in the female genital tract. Mukherjee says, "Interestingly, the rabbit prostate also contains uteroglobin and transglutaminase." In his experiments, sperm taken from the rabbit epididymis, before they had any contact with secretions of the prostate gland, were exposed to immune system cells from a female rabbit. The immune system cells responded as they would to any foreign cells. However, exposure to fluid from the prostate gland or to a mixture of uteroglobin and transglutaminase dramatically suppressed the reaction. Other experiments demonstrated that the uteroglobin was bound to the sperm surface. Whereas other mammals do not produce uteroglobin, they have other uterine proteins that may serve the same function.

"Although these data suggest an antigenic masking role of uteroglobin in combination with transglutaminase *in vitro*, this may not be the only mechanism of nonrejection of the mammalian embryo or the sperm by the mother," Mukherjee says. "It is possible that various other mechanisms for nonrejection ... are sequentially active in fetomaternal relationship during implantation, placentation and gestation in a viviparous animal." Among the other suggested mechanisms are hormonal suppression of the maternal immune system and blockage of maternal antibodies.

Mukherjee suggests that an understanding of the mechanism by which the female mammal tolerates sperm and embryos may aid the development of antifertility drugs and treatments for some forms of infertility. Furthermore, it may provide information about the other obvious example of mammalian tolerance of genetically dissimilar "grafts." Mukherjee predicts, "It may help us understand the mechanism as to how the malignant tumors defy immunological rejection by the affected hosts." —*J. A. Miller*

Fine structure finely measured

The fine structure constant is one of those numbers that physicists are always trying to measure more and more accurately. It is one of the fundamental constants whose values cannot be calculated from theoretical principles but must be determined experimentally. These constants are usually central to important sections of physics, and by a kind of calculational domino effect small variations in, say, the ninth decimal place can have sizable repercussions.

A group of physicists from Bell Laboratories in Murray Hill, N.J. (D. C. Tsui and A. C. Gossard) and the National Bureau of Standards (B. F. Field, M. E. Cage and R. F. Dziuba) report in the Jan. 4 PHYSICAL REVIEW LETTERS that they have used a new technique to determine the fine structure constant to an uncertainty of 0.17 parts per million.

This is not the finest measurement ever made of the constant. That was done at NBS in 1979 by E. R. Williams and P. T. Olsen and goes to 0.11 parts per million. But the new technique means that the two measurements are independent of each other and can be combined to give an even more accurate value, to 0.89 parts per million. According to an NBS announcement, success of the new technique also promises new possibilities for measurement science.

The fine structure constant is the number that measures the strength of electric and magnetic forces relative to other kinds of forces. It thus finds its way into many calculations in electromagnetics, electronics, particle physics, solid-state physics, etc. The measurement technique depends on an electrical resistance phenomenon that the NBS announcement calls the von Klitzing effect, which is a variation on the previously known Hall effect.

If an electric current is flowing in a conductor that happens to lie in a magnetic field, an electric potential will be induced across the conductor in a direction perpendicular to the flow of the original current. This is the so-called Hall potential or Hall voltage. The Hall potential wants to make a current flow across the conductor, and it is opposed by a corresponding resistance, the Hall resistance. The whole effect happens at extremely low temperatures (within a few degrees of absolute zero).

In most substances the Hall voltage and resistance change smoothly as the magnetic field is varied. In 1980, Klaus von Klitzing of the University of Würzburg in West Germany showed that in certain semiconductors the Hall effect is quantized. The Hall voltage and the Hall resistance in these materials change quantally, that is, stepwise, as the magnetic field is varied. The values of the steps can be calculated from an equation involving a few fundamental quantities: the number of the quantum step, the magnetic permeability of a vacuum, the speed of light and the fine structure constant. This opened a way for precise measurement of the fine structure constant independent of previous methods, such as Williams's and Olsen's which used the gyromagnetic properties of protons and the Josephson effect. According to the NBS announcement, by a procedure converse to the measurement of the fine structure constant the quantized resistance feature of the von Klitzing effect could lead to the development of a precise standard for electrical resistance, something that would be an important benefit to precision technology. —*D. Thomsen*

Muscular dystrophy: Promising treatment

Several years ago Scottish and Spanish scientists reported that a drug called allopurinol had helped a limited number of muscular dystrophy patients (SN: 7/19/80, p. 41). Unfortunately, other investigators have not been able to confirm their findings. Now, however, another treatment for muscular dystrophy is looking promising.

There is evidence that the muscle degeneration underlying muscular dystrophy may be initiated by proteinases (enzymes that break down protein). Joanna Hollenberg Sher and colleagues at the State University of New York Downstate Medical Center in Brooklyn, N.Y., found that the proteinase inhibitor leupeptin delayed muscular dystrophy in chickens. They then injected leupeptin dissolved in saline into 16 mice with a genetic susceptibility to muscular dystrophy. Fourteen other mice of the same strain served as one control group and received no treatment, while 10 other mice of the same strain served as a second control group and received only saline. The mice were observed during their first weeks of life for signs of muscular dystrophy-type weakness, then killed so that their muscles could be examined and compared.

As Sher and her co-workers report in the December PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, none of the treated animals showed muscular dystrophy-type weakness, but most of the untreated animals did. What's more, only one of the 16 treated animals experienced muscle degeneration and death, but 19 of the 24 untreated animals did. Some other indications of degeneration were also observed in the muscles of the control animals but not of the treated ones, such as a small diameter in muscle fiber.

In the opinion of Ralph Moss, director of research development for the national headquarters of the Muscular Dystrophy Association in New York City, leupeptin is an "interesting candidate" for the treatment of muscular dystrophy. The reason, he explains, is not just because it has delayed or prevented muscular dystrophy in two animal species, but because of the rationale for trying it in patients in the first place—because it inhibits muscle degeneration. Allopurinol, in contrast, was tried on muscular dystrophy patients only because it prevents the breakdown of adenosine triphosphate (an energy compound necessary for muscle contraction, growth and repair).

Sher and her team will give leupeptin to a handful of muscular dystrophy patients once the Food and Drug Administration approves their clinical trial. Leupeptin is already being tested on muscular dystrophy patients in Japan and is soon to be tested on them in Italy. —*J. A. Treichel*